evidence of CMV infection at post-mortem, eight were receiving steroid therapy (Evans and Williams, 1968). The benefit of steroid therapy in the Guillain-Barré syndrome has yet to be proved (Leading Article, 1975) and, in view of the fatal outcome of the present case, should perhaps be avoided in cases due to CMV infection. Since virological proof of CMV infection only becomes available at a late stage of a patient's illness, earlier evidence should be sought, such as the presence of abnormal liver function tests and abnormal mononuclear cells in the peripheral blood, with a negative Paul Bunnell test.

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Phenobarbital dyskinesia

S. L. LIGHTMAN
M.A., M.R.C.P.

Middlesex Hospital, London W1N 8AA

Summary

A patient was admitted to hospital following an overdose of phenobarbital. During her recovery she developed both chorea and torsion dystonia which could only be related to her high plasma phenobarbital levels.

DIPHENYLHYDANTOIN (phenytoin) overdose is a well recorded cause of chorea and dystonia. A case is now reported of a patient who showed these features after phenobarbital alone. This is thought to be the first report of such an occurrence.

Case report

A 36-year-old nurse was brought by ambulance to the casualty department, having been found semicomatose at a reception centre in central London. She was known to have a personality disorder and

was on treatment with diazepam 15 mg/day and dichloralphenazone 1300 mg at night, prescribed on a daily basis to obviate the danger of overdosage.

On admission she responded in a co-ordinated fashion to painful stimuli but was unresponsive to verbal commands. Her cardiovascular, respiratory and abdominal systems were unremarkable, and in her central nervous system no focal abnormalities could be detected. Her pupils were equal and reacted well to light, she had no doll's eye movements and her fundi were clear. Her reflexes were all present and equal and her plantars flexor.

The following day she was still very drowsy and only arousable by painful stimuli, and it was only 36 hours after admission that she woke up. At this time she was alert and orientated, and while lying in bed she was seen to have choreic movements about the eyes and mouth, and torsion movements at the

shoulder with intorsion of the upper limbs. She also showed athetoid movements of the toes. In addition to these spontaneous movements, examination revealed a dysarthria, phasic nystagmus (more marked to the left owing to an unwillingness to look to the right) and a severe gait ataxia.

The abnormal spontaneous movements gradually decreased over the next 24 hours, and by 72 hours after admission had completely vanished. At this time her nystagmus had also stopped, although her ataxia had now changed in type and was closely related to being observed.

A drug screen was taken at the time of admission and this showed a plasma concentration of 88 mg/litre of phenobarbital (comatose level usually being greater than 75 mg/litre), there was no phenytoin detected, and only a trace of diazepam. On questioning the patient later she admitted that she had stolen the phenobarbital from a friend who was epileptic, and that the only diazepam she had taken was her normal dose (15 mg/day) that she had been taking for several months.

Discussion

It seems unequivocal in this patient that the

chorea and torsion dystonia noted in the recovery phase from her phenobarbital overdose were directly related to toxic levels of this drug. It is of course well recognized that anticonvulsants, when taken in excess, are able to produce a variety of dyskinesias. Chorea and dystonia, however, have only been recorded in patients who have grossly elevated serum levels of phenytoin, and have never been reported with other anticonvulsants alone. A recent review (Chadwick, Reynolds and Marsden, 1976) states categorically that 'diphenylhydantoin, but not other anticonvulsants, may cause orofacial dyskinesias, limb chorea and dystonia in intoxicated patients'. The present case demonstrates that both chorea and torsion dystonia may be caused by phenobarbital alone.

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Cerebral haemorrhage in haemophilia

MICHAEL BENNETT*
M.B., B.S., M.R.C.P.

JOHN A. SILLS
M.B., B.Chir., M.R.C.P., D.C.H.

The Royal Infirmary and Royal Hospital for Sick Children, Edinburgh

Summary

A young male haemophiliac child is here described in whom intracerebral haemorrhage required craniotomy and the subsequent insertion of a ventriculo-peritoneal shunt. These procedures can now be attempted in severe haemophilia A because of the ease in clotting factor replacement therapy.

Introduction

The occurrence of a cerebral haemorrhage in a haemophiliac has nearly always a fatal outcome (Silverstein, 1960). Recently, however, there has been a rapid advance in the treatment of haemophilia

with the development of potent small volume human factor VIII replacement therapy. Here, now, is described how such modern therapy was applied to a haemophiliac with cerebral haemorrhage in an attempt to improve the prognosis.

Case report

The patient was diagnosed as having haemophilia A at the age of nine months. Bruising had been noticed during the preceding two months. The biological factor VIII level was 2%, factor IX level was 96% and the bleeding time (Ivy) was normal at 3 min. The platelet count was $480 \times 10^9/l$ and the prothrombin time was normal. There was no family history of a bleeding diathesis. Over the next three

* Present address: Department of Haematology, The Royal Free Hospital, London NW3 2QG.